EXHIBIT 2

THE UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF DELAWARE

NIPPON SHINYAKU CO., LTD., Plaintiff, v. SAREPTA THERAPEUTICS, INC., Defendant.	C.A. No. 21-1015 (JLH)
SAREPTA THERAPEUTICS, INC. and THE UNIVERSITY OF WESTERN AUSTRALIA, Defendant/Counter-Plaintiffs, v. NIPPON SHINYAKU CO., LTD. and NS PHARMA, INC. Plaintiff/Counter-Defendants.	

EXHIBIT 2

PARTIES' STATEMENT OF UNCONTESTED FACTS

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I. THE PARTIES AND THE NATURE OF THE ACTION[1]

A. The Parties

1. Sarepta is the Defendant and a Counter-Plaintiff in this action. Sarepta is a corporation organized and existing under the laws of the State of Delaware with a principal place of business located at 215 First Street, Cambridge, Massachusetts. Sarepta has licensed certain patents assigned to UWA, including the '851 Patent (the "Wilton Patent") asserted against Nippon Shinyaku and NS Pharma in this action. Sarepta markets VYONDYS 53® (golodirsen) in the United States.

2. UWA is a Counter-Plaintiff in this action. UWA is a public research university organized and existing under the laws of Australia with its main campus and offices located at 35 Stirling Highway, Crawley, Perth, Western Australia 6009. UWA is the assignee and licensor of the Wilton Patent.

3. Nippon Shinyaku is the Plaintiff and a Counter-Defendant in this action. Nippon Shinyaku is a Japanese company with a principal place of business at 14, Nishinosho-Monguchi-cho, Kisshoin, Minami-ku, Kyoto 601-8550, Japan. Nippon Shinyaku has asserted the '092 Patent, the '461 Patent, the '106 Patent, the '741 Patent, and the '217 Patent against Sarepta.

4. NS Pharma is a Counter-Defendant in this action. NS Pharma is a corporation organized and existing under the laws of the State of Delaware with its principal place of business at 140 East Ridgewood Ave., Suite 280S, Paramus, NJ 07652. NS Pharma is a wholly owned U.S. subsidiary of Nippon Shinyaku. NS Pharma is Nippon Shinyaku's U.S. Agent authorized by the FDA to market VILTEPSO ® (viltolarsen) in the United States.

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[1] Headings are included for the convenience of the Court only. The parties acknowledge and agree insofar as any facts are read to the jury that headings shall not be read to the jury.

5. Nippon Shinyaku and NS Pharma have entered into an exclusive "License and Supply" agreement whereby Nippon Shinyaku granted rights to NS Pharma to: (1) import and purchase VILTEPSO® (viltolarsen) from Nippon Shinyaku; (2) package VILTEPSO® (viltolarsen) for sale in the United States; and (3) export, sell, distribute, and promote VILTEPSO® (viltolarsen) in the United States.

6. Sarepta and Nippon Shinyaku each develop and commercialize exon 53 skipping therapies for the treatment of DMD in patients amenable to exon 53 skipping in the United States.

7. Aside from Sarepta and Nippon Shinyaku, other companies have attempted to develop a commercial exon skipping ASO product targeting exon 53, BMN053, but have failed to do so.

8. Sarepta and Nippon Shinyaku are the only companies with FDA clearance to market oligonucleotide therapies that induce exon 53-skipping for the treatment of DMD for patients in need thereof.

9. Sarepta's product is marketed under the name VYONDYS 53®, and Nippon Shinyaku's product is marketed under the name VILTEPSO®.

II. PATENTS-IN-SUIT

A. The Wilton Patent

10. Sarepta controlled prosecution of the Wilton Patent before the USPTO.

11. Sarepta provided input to outside counsel prosecuting the Wilton Patent before the USPTO.

12. Sarepta provided input regarding the arguments and evidence from UWA during the interference proceedings before the USPTO.

13. The Wilton Patent is directed towards antisense oligonucleotide-based therapies for the treatment of DMD.

14. The Wilton Patent is titled: "ANTISENSE OLIGONUCLEOTIDES FOR INDUCING EXON SKIPPING AND METHODS OF USE THEREOF."

15. The Wilton Patent claims priority to the '943 PCT Application, filed June 28, 2005.

16. The specification of the Wilton Patent is substantively identical to the specification of the '943 PCT Application. The effective filing date of the Wilton Patent is June 28, 2005.

17. The Wilton Patent stems from the '172 Application, filed September 14, 2017.

18. The Wilton Patent issued on June 12, 2018.

19. The Wilton Patent is set to expire on June 28, 2025.

20. The Wilton Patent lists Stephen Donald Wilton, Sue Fletcher, and Graham McClorey as inventors.

21. The Wilton Patent is assigned to UWA.

22. Sarepta has exclusively licensed the Wilton Patent from UWA.

23. On June 12, 2018, the '851 Patent entitled "Antisense Oligonucleotides for Inducing Exon Skipping and Methods of Use Thereof" issued to UWA as assignee with named inventors Stephen Donald Wilton, Sue Fletcher, and Graham McClorey.

24. Sarepta has listed the Wilton Patent on its FDA Orange Book listing for VYONDYS 53®.

25. The FDA Orange Book entry for Exondys51® (eteplirsen) identifies one or more patents claiming priority to PCT/AU2005/000943.

26. The Wilton Patent claim encompasses Sarepta's VYONDYS 53®.

27. The Wilton Patent claim encompasses VILTEPSO®.

28. There was a long-felt but unmet need for an effective and safe treatment of DMD patients before the effective filing date of the Wilton Patent.

29. The claimed inventions of the Wilton Patent satisfied a long-felt but unmet need for patients amenable to exon 53 skipping treatment that existed before the effective filing date of the Wilton Patent.

30. Before the Wilton Patent was filed in June 2005, there was no known specific region within exon 53 of the human dystrophin pre-mRNA that researchers could target with ASOs to induce exon skipping.

31. Nippon Shinyaku and NS Pharma were aware of the Wilton Patent by at least September 2019.

32. For the Wilton Patent,[2] a person of ordinary skill in the art ("POSA") is an individual that has an M.D., Ph.D., or lower degree with expertise in molecular biology, biochemistry or a related area, and experience with neuromuscular or genetic diseases and/or designing and testing antisense oligonucleotides for splice-site switching/exon skipping applications. The POSA would have general knowledge of antisense oligonucleotide chemical modifications to the backbone, nucleobases and other manipulations that can alter the activity of the antisense molecule, as well as delivery methods for antisense oligonucleotides. A POSA would also have general knowledge regarding using antisense oligonucleotides in cell-free, cell-based and/or in vivo experiments, as well as DMD models and the use of antisense oligonucleotides to induce skipping of DMD exons to correct the open reading frame of the RNA transcripts.

B. The NS Patents

33. The '092 Patent stems from the '213 Application, filed March 20, 2019. The '092 Patent issued on August 20, 2019.

[2] Given that both parties' experts agree that the definition of a person of ordinary skill in the art does not alter their analyses and the parties agree that they will not raise arguments or question witnesses (e.g., by cross-examination) regarding the differences between the competing definitions, Nippon Shinyaku's definition can be the instruction to the jury.

34. The ’461 Patent stems from the ’451 Application, filed March 26, 2019. The ’461 Patent issued on September 10, 2019.

35. The ’106 Patent stems from the ’427 Application, filed March 29, 2019. The ’106 Patent issued on November 26, 2019.

36. The ’741 Patent stems from the ’537 Application, filed June 24, 2019. The ’741 Patent issued on May 12, 2020.

37. The ’217 Patent stems from the ’686 Application, filed December 12, 2019. The ’217 Patent issued on May 26, 2020.

38. Each of the NS Patents is titled: “ANTISENSE NUCLEIC ACIDS.”

39. Each of the NS Patents claims priority to U.S. Patent No. 9,708,361, which in turn claims priority to the ’318 PCT Application, filed August 31, 2011. The specifications of the NS Patents are substantively identical to each other and to the specification of the ’318 PCT Application. The effective filing date of the NS Patents is August 31, 2011.

40. Each of the NS Patents is set to expire on August 31, 2031.

41. Each of the NS Patents lists Naoki Watanabe, Youhei Satou, Shin’ichi Takeda, and Tetsuya Nagata as inventors.

42. Each of the NS Patents lists Nippon Shinyaku and National Center of Neurology and Psychiatry as assignees.

43. On August 20, 2019, the ’092 Patent, entitled “Antisense Nucleic Acids,” issued to Nippon Shinyaku and NCNP as assignees with named inventors Naoki Watanabe, Youhei Satou, Shin’ichi Takeda, and Tetsuya Nagata.

44. On September 10, 2019, the '461 Patent entitled "Antisense Nucleic Acids," issued to Nippon Shinyaku and NCNP as assignees with named inventors Naoki Watanabe, Youhei Satou, Shin'ichi Takeda, and Tetsuya Nagata.

45. On November 26, 2019, the '106 Patent entitled "Antisense Nucleic Acids," issued to Nippon Shinyaku and NCNP as assignees with named inventors Naoki Watanabe, Youhei Satou, Shin'ichi Takeda, and Tetsuya Nagata.

46. On May 12, 2020, the '741 Patent entitled "Antisense Nucleic Acids," issued to Nippon Shinyaku and NCNP as assignees with named inventors Naoki Watanabe, Youhei Satou, Shin'ichi Takeda, and Tetsuya Nagata.

47. On May 26, 2020, the '217 Patent entitled "Antisense Nucleic Acids," issued to Nippon Shinyaku and NCNP as assignees with named inventors Naoki Watanabe, Youhei Satou, Shin'ichi Takeda, and Tetsuya Nagata.

48. Each of the NS Patent asserted claims encompasses Sarepta's VYONDYS 53®.

49. The Sazani 2010 paper was not submitted to the USPTO during prosecution of the NS Patents.

50. At least one of the Inventors of the NS Patents was aware of the Sazani 2010 paper prior to the July 18, 2017 issue date of the '361 Patent.

51. Test results additional to what was disclosed in the specification show that SEQ ID NO: 57, corresponding to positions 36 to 60, did not exhibit superior skipping to the oligonucleotide corresponding to positions 30 to 59, taught as providing the highest activity by the Popplewell 2010 paper.

52. Prior to August 31, 2011, the Inventors of the NS Patents did not perform the actual chemical synthesis of a PMO that has 25 bases and contains a sequence of bases that is 100% complementary to positions +36+60 of Exon 53.

53. Prior to August 31, 2011, the others instructed by the Inventors of the NS Patents to synthesize a PMO that has 25 bases and contains a sequence of bases that is 100% complementary to positions +36+60 of Exon 53 had not completed such a synthesis.

54. The NS Patents do not disclose data derived from the experimental testing of golodirsen.

55. For the NS Patents,[3] a person of ordinary skill in the art ("POSA") is an individual that has an M.D., Ph.D., or lower degree with expertise in molecular biology, biochemistry or a related area, and experience with neuromuscular or genetic diseases and/or designing and testing antisense oligonucleotides for splice-site switching/exon skipping applications. The POSA would have general knowledge of antisense oligonucleotide chemical modifications to the backbone, nucleobases and other manipulations that can alter the activity of the antisense molecule, as well as delivery methods for antisense oligonucleotides. A POSA would also have general knowledge regarding using antisense oligonucleotides in cell-free, cell-based and/or in vivo experiments, as well as DMD models and the use of antisense oligonucleotides to induce skipping of DMD exons to correct the open reading frame of the RNA transcripts.

[3] Given that both parties' experts agree that the definition of a person of ordinary skill in the art does not alter their analyses and the parties agree that they will not raise arguments or question witnesses (e.g., by cross-examination) regarding the differences between the competing definitions, NS's definition can be the instruction to the jury.

III. PRODUCTS AT ISSUE

A. DMD

56. DMD is a severe X chromosome-linked genetic disorder that predominantly affects young boys. DMD occurs in about one out of every 3,600 male infants worldwide and is the most common type of muscular dystrophy.

57. Children with DMD typically suffer muscle weakness between ages three to five and progressively lose muscle function and quality-of-life. Children with DMD progressively lose the ability to perform everyday activities and often require a wheelchair and assistance by their early teens. As DMD progresses, life-threatening heart and respiratory conditions can occur. Although disease severity and life expectancy vary, patients with DMD typically die of the disease in their 20s or 30s.

58. DMD is caused by a lack of functional dystrophin—a protein that maintains the integrity of muscle fibers. Normally, dystrophin is encoded by the dystrophin gene (also referred to as the *DMD* gene). Patients with DMD lack functional dystrophin because the dystrophin gene contains a mutation that results in premature termination of dystrophin production.

59. There is currently no known cure for DMD.

B. Sarepta's VYONDYS 53® (Golodirsen) Product

60. Sarepta sought and received FDA approval of eteplirsen before golodirsen.

61. On May 22, 2018, FDA granted Orphan Drug Designation to Sarepta for its antisense oligonucleotide-based therapy, golodirsen, which would eventually be approved and marketed under the name VYONDYS 53®.

62. Sarepta's VYONDYS 53® (golodirsen) obtained accelerated approval from the FDA on December 12, 2019. VYONDYS 53® (golodirsen) is indicated for the treatment of DMD in patients who have a confirmed mutation in the DMD gene that is amenable to exon 53 skipping.

VYONDYS 53® (golodirsen) is the first-ever FDA-approved treatment indicated for patients amenable to exon 53 skipping in the United States.

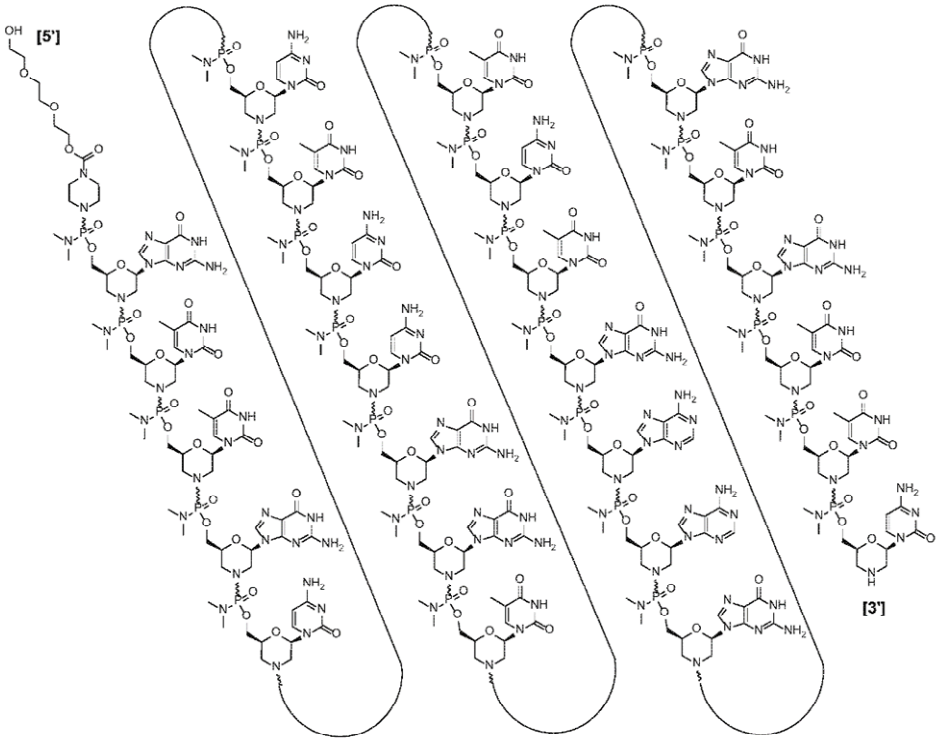
63. Sarepta sponsored clinical trials in the United States for VYONDYS 53®.

64. Following FDA approval, Sarepta has marketed, offered to sell, and/or sold VYONDYS 53® in the United States for the treatment of DMD patients who have a confirmed mutation of the dystrophin gene that is amenable to exon 53 skipping.

65. VYONDYS 53® is administered to patients in the United States.

66. The active ingredient of VYONDYS 53® (golodirsen) is golodirsen. Golodirsen is an antisense oligonucleotide. The 25-base sequence of golodirsen from the 5'-end to the 3'-end is GTTGCCTCCGGTTCTGAAGGTGTTC.

67. The structure and 25-base sequence of golodirsen are shown below:



68. Golodirsen is 25 bases in length.

69. Golodirsen targets a region within exon 53 of the human dystrophin pre-mRNA. Specifically, the target region to which golodirsen binds corresponds to positions 36 to 60 of exon 53 of the human dystrophin pre-mRNA.

70. The target region to which golodirsen binds is within positions 23 to 69 of exon 53 of the human dystrophin pre-mRNA.

71. The 25-base sequence of golodirsen is 100% complementary to consecutive bases of its target region of exon 53 of the human dystrophin pre-mRNA. Specifically, the 25-base sequence of golodirsen is 100% complementary to positions 36 to 60 of exon 53 of the human dystrophin pre-mRNA.

72. The 25-base sequence of golodirsen comprises at least 12 consecutive bases of CUG AAG GUG UUC UUG UAC UUC AUC C, but having thymine bases instead of uracil bases.

73. Golodirsen contains adenine (A), thymine (T), guanine (G), and cytosine (C). Golodirsen does not contain uracil (U).

74. Golodirsen is a phosphorodiamidate morpholino oligomer.

75. Golodirsen is designed to bind to exon 53 of the human dystrophin pre-mRNA and results in exclusion of this exon during mRNA processing in patients with genetic mutations that are amenable to exon 53 skipping.

76. When appropriately delivered into human cells, golodirsen induces exon 53 skipping.

77. VYONDYS 53® (golodirsen) is supplied in single-dose vials containing 100 mg/2 mL golodirsen in isotonic phosphate buffered saline solution.

78. VYONDYS 53® (golodirsen) is administered intravenously.

79. Sarepta's label for VYONDYS 53® provides that "VYONDYS 53 is administered via intravenous infusion."

80. The active pharmaceutical ingredient in VYONDYS 53® is referred to and known as golodirsen.

81. The structure of golodirsen is set forth in Section 11 of the Prescribing Information.

82. Golodirsen is an antisense oligonucleotide of the phosphorodiamidate morpholino oligomer (PMO) subclass that contains 25 linked subunits and is designed to bind to exon 53 of human dystrophin pre-RNA.

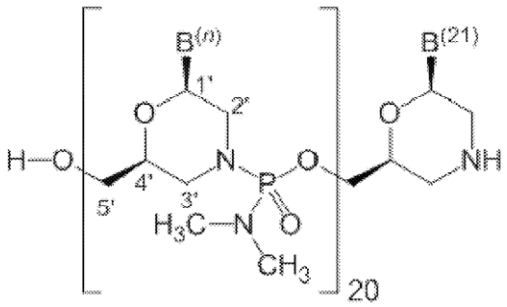
83. The sequence of bases from the 5' end to 3' end of golodirsen is GTTGCCTCCGGTTCTGAAGGTGTTC.

C. NS's VILTEPSO® (Viltolarsen) Product

84. NS's VILTEPSO® (viltolarsen) obtained accelerated approval from the FDA on August 12, 2020. VILTEPSO® (viltolarsen) is indicated for the treatment of DMD in patients who have a confirmed mutation in the DMD gene that is amenable to exon 53 skipping. VILTEPSO® (viltolarsen) is the second FDA-approved treatment indicated for patients amenable to exon 53 skipping in the United States.

85. The active ingredient of VILTEPSO® (viltolarsen) is viltolarsen. Viltolarsen is an antisense oligonucleotide. The 21-base sequence of viltolarsen from the 5'-end to the 3'-end is CCTCCGGTTCTGAAGGTGTTC.

86. The structure and 21-base sequence of viltolarsen are shown below:



CCTCCGGTTC TGAAGGTGTT C

87. Viltolarsen is 21 bases in length.

88. Viltolarsen contains a hydroxyl (OH) group at its 5' end.

89. Viltolarsen targets a region within exon 53 of the human dystrophin pre-mRNA. Specifically, the target region to which viltolarsen binds corresponds to positions 36 to 56 of exon 53 of the human dystrophin pre-mRNA.

90. The target region to which viltolarsen binds is within positions 23 to 69 of exon 53 of the human dystrophin pre-mRNA.

91. The 21-base sequence of viltolarsen is 100% complementary to consecutive bases of its target region of exon 53 of the human dystrophin pre-mRNA. Specifically, the 21-base sequence of viltolarsen is 100% complementary to positions 36 to 56 of exon 53 of the human dystrophin pre-mRNA.

92. The 21-base sequence of viltolarsen comprises at least 12 consecutive bases of CUG AAG GUG UUC UUG UAC UUC AUC C, but having thymine bases instead of uracil bases.

93. Viltolarsen contains adenine (A), thymine (T), guanine (G), and cytosine (C). Viltolarsen does not contain uracil (U).

94. Viltolarsen is a phosphorodiamidate morpholino oligomer.

95. Viltolarsen is designed to bind to exon 53 of the human dystrophin pre-mRNA and results in exclusion of this exon during mRNA processing in patients with genetic mutations that are amenable to exon 53 skipping.

96. When appropriately delivered into human cells, viltolarsen induces exon 53 skipping.

97. VILTEPSO® (viltolarsen) is supplied in single-dose vials containing 250 mg/5 mL viltolarsen in 0.9% sodium chloride.

98. VILTEPSO® (viltolarsen) is administered intravenously.

99. Nippon Shinyaku directly or through its agents and other third parties manufactures, markets, offers to sell, sells, and/or distributes VILTEPSO® (viltolarsen) in the United States.

100. Nippon Shinyaku is the sole supplier of VILTEPSO® (viltolarsen) to the United States and sells VILTEPSO® (viltolarsen) to NS Pharma, with title passing after importation upon delivery to a Rockford, Illinois facility.

101. NS Pharma offers to sell and sells VILTEPSO® (viltolarsen) in the United States.

102. On January 16, 2017, FDA granted Orphan Drug Designation to Nippon Shinyaku for its antisense oligonucleotide-based therapy that would eventually be approved and marketed under the name VILTEPSO®.

103. On August 12, 2020, the FDA granted accelerated approval to VILTEPSO® injection for the treatment of DMD in patients who have a confirmed mutation of the DMD gene that is amenable to exon 53 skipping.

104. Nippon Shinyaku sells units of VILTEPSO® to NS Pharma.

105. NS Pharma sells VILTEPSO® in the United States.

106. Nippon Shinyaku sells units of VILTEPSO ® to NS Pharma for re-sale in the United States pursuant to a License and Supply Agreement (effective April 1, 2020).

107. NS Pharma re-sells units of VILTEPSO® that it purchases from Nippon Shinyaku.

108. Title transfers to NS Pharma upon the units' delivery to a Rockford, Illinois facility.

109. The active pharmaceutical ingredient in VILTEPSO® is referred to and known as viltolarsen.

110. Viltolarsen is an antisense oligonucleotide of the PMO subclass.

111. Viltolarsen contains 21 linked subunits and is designed to bind to exon 53 of human dystrophin pre-RNA.

112. The sequence of bases of viltolarsen from the 5' end to the 3' end is CCTCCGGTTCTGAAGGTGTTC.

113. Viltolarsen is designed to bind to exon 53 of dystrophin pre-mRNA resulting in exclusion of this exon during mRNA processing in patients with genetic mutations that are amenable to exon 53 skipping.

114. Viltolarsen contains thymine bases in place of uracil bases.